

GN

Cardio: Infective endo

Chest : good pasture

Blood: Henoch schonlein

fever: Extra hepatic of viral hepatitis

Rh : e SLE

Introduction

Q: normal

* Kidney Functions :-

- ① Acts as filter system (Glomerular filtration) (1000000 nephron in each Kid)
- ② Gets rid of waste products of metabolism eg: uric acid } of ptn
urea }
- ③ Homeostasis: control internal environment
- eg: drugs }
as digoxin }
- ① - Acid base balance as in eg: COPD
↳ CO_2 retention (acidosis)
↓
Kidney reabsorbs HCO_3^-
- ② - Body fluid balance as in eg: H_2O retention
- ③ - Blood pressure control
- if hypotension
→ rennin →
↑ aldosterone
→ salt & H_2O retention
- if hypertension
→ ADH ↓ → ↑ water loss
- if hypotension
→ ADH ↑ → ↑ water retention
- ④ Concentration of electrolytes (esp. K^+)
- digitoxin
liver nephron

④ Hormones (endocrinal)

- 1. **Renin** : control BP
- 2. **Erythropoietin** : to ++ RBCs formation in BM
- 3. **Active vit D** : to maintain healthy bones
- 4. **Urodilatin** : in response to $\left\{ \begin{array}{l} \text{HTN} \\ \text{hypervolemia} \end{array} \right. \rightarrow \text{Diuresis}$
 mechanism of diuresis: by $\uparrow \text{RBF} \rightarrow$

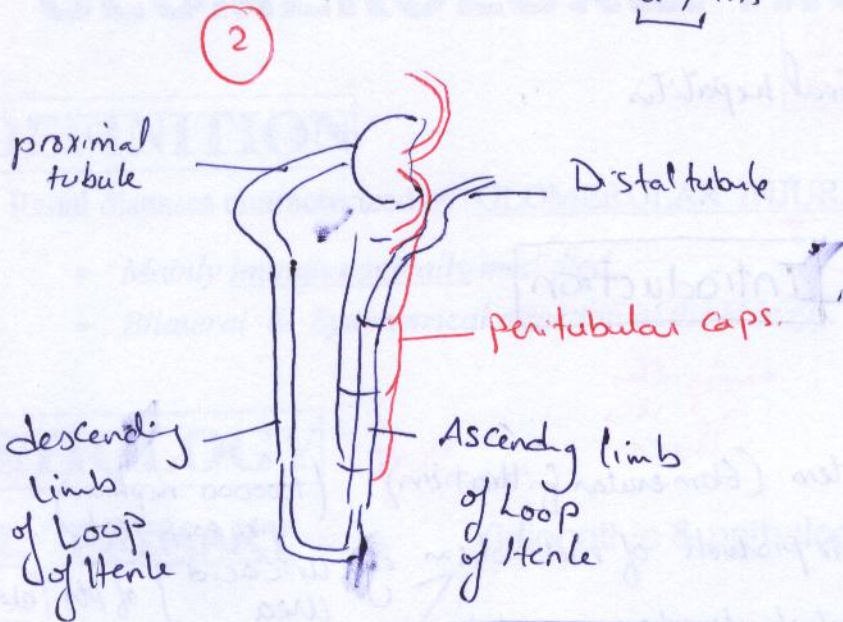
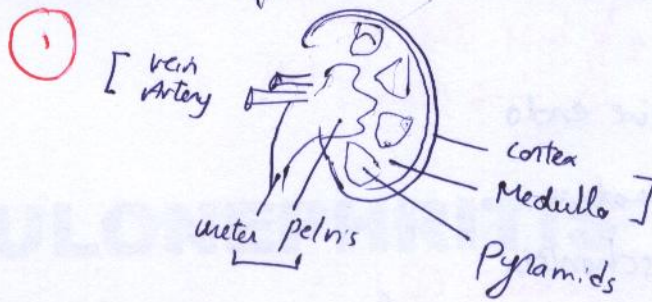
↓K only in

- TN
- nephrotic

- also
 - digitalis
 - dopamine

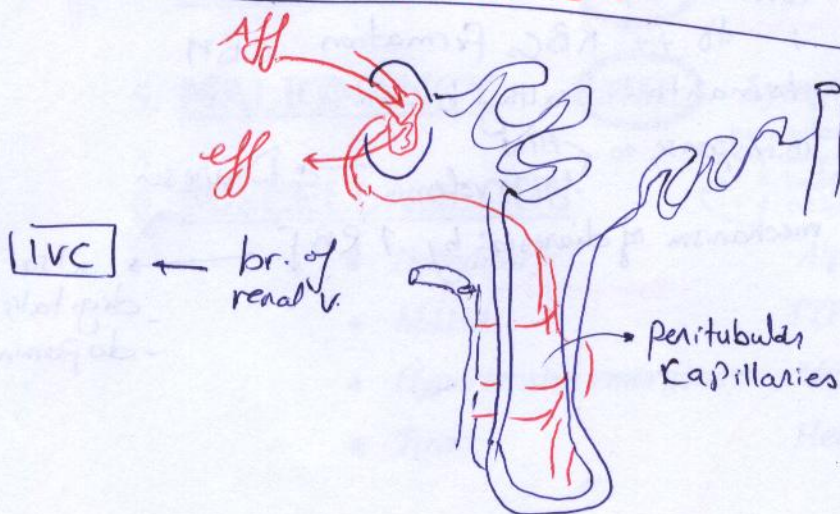
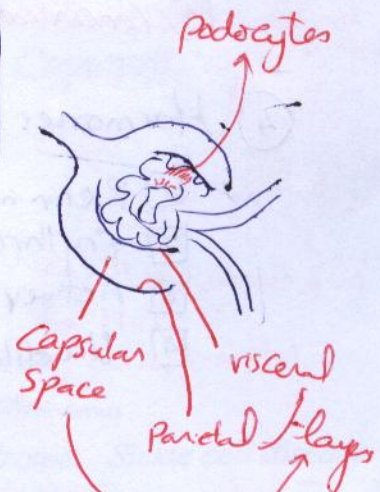
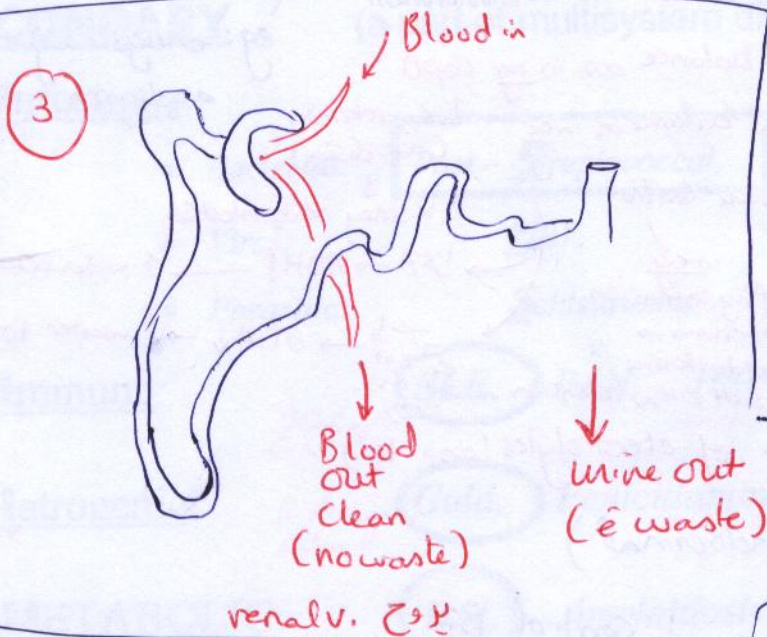
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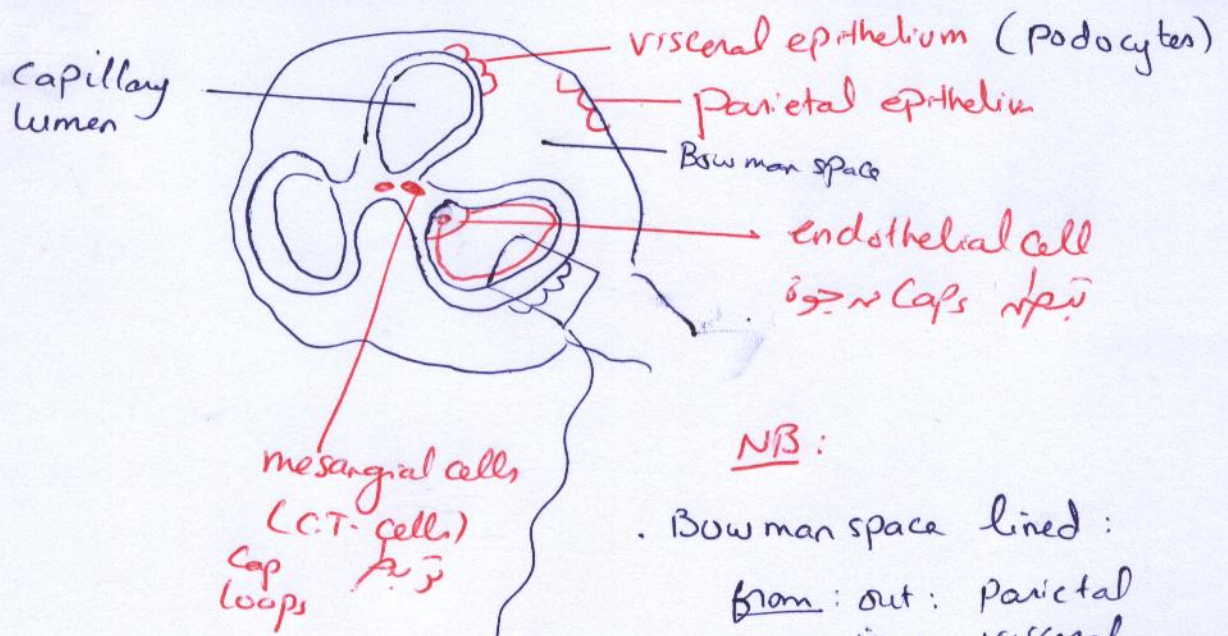
LANZAROTE
Caliente.COM



الترتيب كالتالي

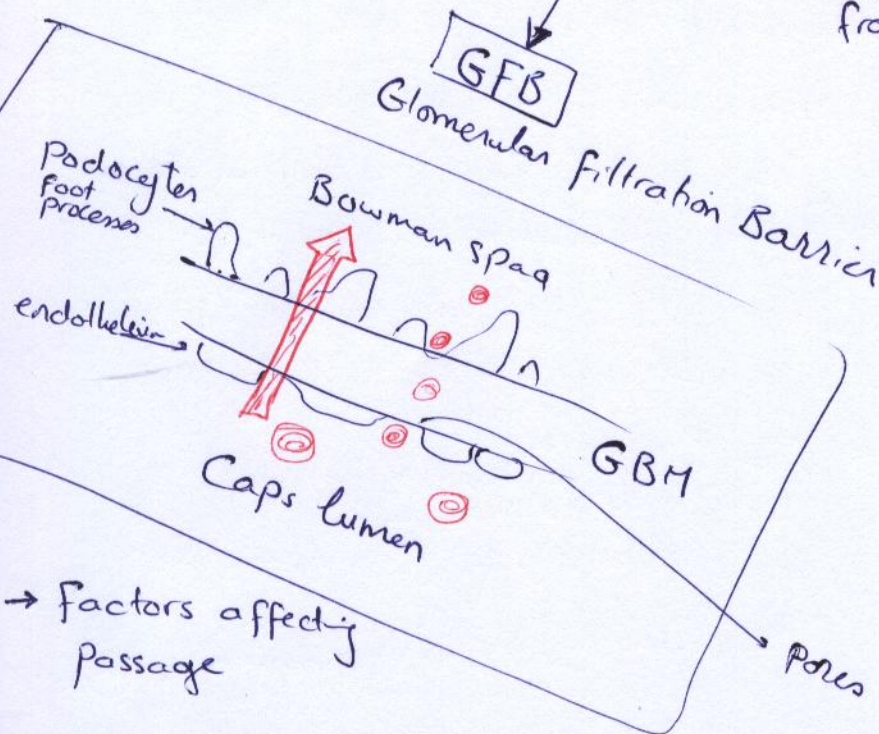
Renal A.
 Aff
 Glomerulus & p.t.
 eff
 peritubular caps.
 Br. of renal v.
 renal v.





NB:

- Bowman space lined:
from out: parietal
in: visceral
- cap lumen lined
from out: visceral
in: endothelium



→ Factors affecting passage

① size of particles (acc. to pores size)

② charges — neutral only pass
— -ve not pass (as podocytes & endothelium are -ve → repulsion)

NB • ptn not pass as large
— -ve charge

• if podocytes destroyed → ptn pass

• ptn traces may pass thru 2 barriers

overcome size → reabsorbed by kidney
change
0-150 mg/24h overcome also the reabs. by tubule

لأنه كان يمكنه نقل البروتينات، لم
ونزل البروتين في لumen

WhiteKnightLove

Oral ^{100%}

- infections
- emergency

مرحبا و Kid في
لنرى القصة

1

GLOMERULONEPHRITIS (GN)

Q causes
Q Classification

DEFINITION

- Renal diseases characterized by GLOMERULAR INJURY & INFLAMMATION:

- Mainly immunologically mediated.
- Bilateral & Symmetrical affection of the kidneys.

معيشة
GN
من كل واحد

Ab's
الأمراض
المناعية

ETIOLOGY

ways

I. PRIMARY

(Idiopathic & pathology is confined to kidney)

II. SECONDARY

(a part of multisystem disorder)

1. Infections:

- Bacterial: Post-Streptococcal, SBE, Syphilis.
- Viral: HBV, HCV, HIV.
- Parasitic: Schistosoma. (not common)

imm. complex (Ag + Ab + complement)

2. Immune:

SLE, PAN, HSP, GPS, EMC.
90% GN → SLE
henoch → HSP
Good's Pasture syndrome (lung) → GPS
Essential Mixed Cryoglobulinemia → EMC

3. Iatrogenic:

Gold, Penicillamine, Captopril.
R.A. Asthma → Gold
Wilson's scleroderma R.A. → Penicillamine
(ACEI short) → Captopril

4. METABOLIC:

DM, Amyloidosis.
Kliemsteil Wilson → DM

5. MALIGNANCY:

MM, Lymphoma.
Kidney cancer → MM
hereditary
1 Congenital damage glomeruli
2 Deafness
3 Ocular defect: ant. lenticulus

6. MISCELLANEOUS:

- Hereditary: Alport's syndrome, Sickle cell disease.
- MAHA: TTP, HUS, DIC.
Microangiopathic → MAHA
crisis of B.V. → HUS
- Hypertensive emergency: Malignant hypertension.
- Toxic: Heroin, Mercury.
spleen bone Kid → Malignant hypertension

emergency = target organ damage

1

PATHOGENESIS

VIP

Sometimes by both imm & non imm = infections

1. IMMUNOLOGICAL INJURY:

الاعراض

- Immune complexes: most types of GN, e.g. PSGN, SBE, SLE.
- Anti - GBM antibodies: some types of GN, e.g. GPS.
- Abnormal complement activation: some cases of idiopathic GN.

→ only complement is activated
مثلا، في
eg: PNH

2. NON - IMMUNOLOGICAL INJURY:

A. Accumulation of abnormal metabolites: e.g.

- DM. ————— Sorbitol
- Amyloidosis. ————— O₂ free radicals

B. High intraglomerular pressure: e.g.

- Hypertensive emergency: Malignant hypertension.

C. Intravascular coagulation:

- TTP, HUS, DIC.

oral

- ① ischemic necrosis of glomerulus
- ② endothelial injury (rough)

D. Deposition diseases:

- EMC. ————— Cryoglobulins
- Amyloidosis. ————— amyloid ptn
- MM. ————— Bence - jones ptn

- In these diseases, abnormal proteins are deposited in the glomeruli → inflammatory reaction and / or glomerulosclerosis.

E. Toxic injury: directly

- Heroin, Mercury.

F. INFECTION:

(Multifactorial) < imm non imm

- Direct infection of renal cells. (organism)
- Release of nephrotoxins: e.g. E - coli → HUS.
- Immune complexes: e.g. PSGN, SBE.
- Chronic stimulation of amyloid formation.

مثلا
مثلا

esp TB
Bronchiectasis
lung abscess

CLASSIFICATION

1. Etiology:

[of glomerular injury]

- Primary: idiopathic.
- Secondary: a part of multisystem disorder.

2. Course:

[of glomerular injury]

- Acute: over days to weeks.
- Subacute (*rapidly progressive*): over weeks to months.
- Chronic: *أسرع بمرور* over months to years.

3. Distribution:

[of glomerular injury]

a) In the kidney:

- Diffuse: Majority of glomeruli (> 50 %).
- Focal: Minority of glomeruli (< 50 %).

b) In the glomerulus:

- Global: affects *almost all* of the glomerulus.
- Segmental: affects *only some parts* of the glomerulus.

4. Pathology:

[of glomerular injury]

- a) Non - Proliferative = "no increase in glomerular cell number"

Non - Proliferative GN

1. Diffuse: *>50* Minimal change GN, Membranous GN.
2. Focal: *<50* Focal segmental glomerulosclerosis.

- b) Proliferative = "increase in glomerular cell number" ... Why ??

1. Infiltration of: glomerulus by inflammatory cells (neutrophils & monocytes).

2. Proliferation of: resident glomerular cells:

- Intracapillary proliferation: endothelial or mesangial cells.
- Extracapillary proliferation: epithelial cells in Bowman's space.

Proliferative GN

1. Diffuse: *>50*
 - Membrano - proliferative GN.
 - Mesangio - proliferative GN.
 - Crescentic GN.
 - Diffuse - proliferative GN.
2. Focal: *<50* IgA nephropathy (Berger's disease).

كده
ولا كده
Bilateral
& symmetrical

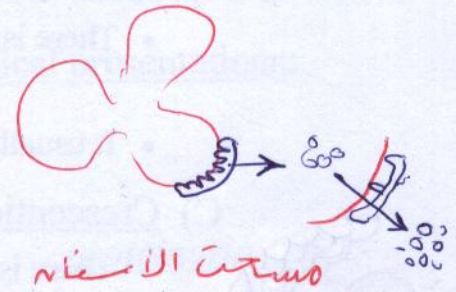
as
injury
& inflammation
↓
cytokine

visceral
parietal

PATHOLOGY

I. Non - Proliferative GN:

1. Diffuse: >50%



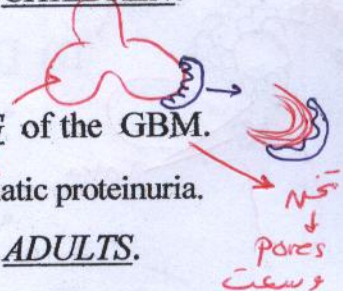
A) Minimal change GN:

- Light microscope: No abnormality.
- Electron microscope: Diffuse EFFACEMENT of the foot processes of the PODOCYTES.
 no narrow pores
 no -ve charge
- It usually presents by: Nephrotic syndrome. heavy pturia
- It is the most common form of Nephrotic syndrome in: CHILDREN.

oral
 ناتي بياض
 nephrotic
 and
 effacement

B) Membranous GN:

- There is: Diffuse LEAKY THICKENING of the GBM.
- It usually presents by: Nephrotic syndrome ^{severe} or asymptomatic proteinuria. ^{mild}
- It is the most common form of Nephrotic syndrome in: ADULTS.



nephrotic
 ن

2. Focal: (FSGS) "Focal segmental glomerulosclerosis"

- There is: sclerosis (fibrosis & scarring) & hyalinosis.
- Focal sclerosis: Kid involves Minority of glomeruli (< 50 %).
- Segmental sclerosis: Glomerulus involves only some parts of the glomerulus.
- It usually presents by: Nephrotic syndrome.
- It causes Nephrotic syndrome in: CHILDREN & ADULTS.



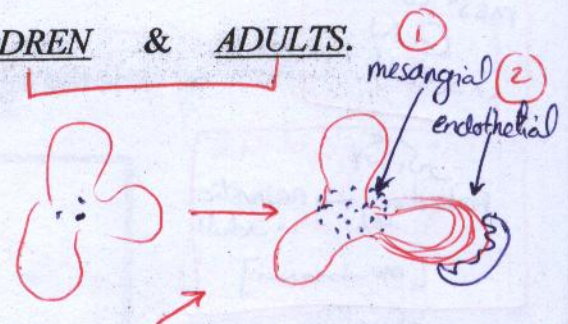
→ damage
 of
 GBM

II. Proliferative GN:

1. Diffuse:

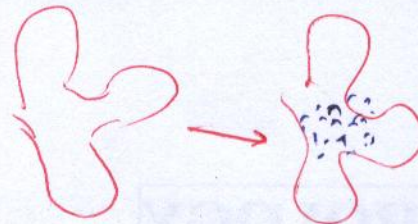
A) Membrano - proliferative GN:

- There is: Diffuse LEAKY THICKENING of the GBM.
- There is: Intracapillary Proliferation → obs. of Cap. lumen [Mesangial cells, Matrix & Endothelial cells].
- It usually presents by: Nephrotic syndrome (or) Nephritic syndrome.



الوحدة
 proliferative
 nephrotic (↑pturia)

All Prolif. → nephritic except Crescentic
 All Prolif → eout nephritic except membrano prolif.
 = nephritic = All non prolif + membr prolif.
 = nephritic = All prolif except Crescentic



5

B) Mesangio – proliferative GN:

- There is: Marked Proliferation of Mesangial cells and Matrix → encroachment on capillary lumen.
- It usually presents by: Nephritic syndrome. (↓ GFR)

C) Crescentic GN (RPGN)

- There is: Extracapillary Proliferation (marked & rapid) [Parietal epithelial cells in Bowman's space]
- There is: مترقوا على caps Marked Epithelial crescent formation → → →
 - encroachment on capillary lumen
 - reduction of Bowman's space

- It usually presents by: Rapidly progressive RF (over weeks to months).

D) Diffuse – proliferative GN:

- There is: Intracapillary & Extracapillary Proliferation Endothelial, Mesangial & Epithelial cells.
- It usually presents by: Nephritic syndrome & ARF (over days to weeks).

2. Focal:

“IgA nephropathy or Berger's disease”

- There is: mesangial proliferation, glomerular inflammation & necrosis.
- Focal affection: involves **Minority** of glomeruli (< 50 %).
- Segmental affection: involves **only some parts** of the glomerulus.
- It usually presents by: Hematuria, mild to moderate ↓GFR, Nephritic syndrome.
- It is the most common type of GN world – wide in: ADULTS.

Chronic GN

- It is a chronic form characterized by:
 - Glomerular: atrophy.
 - Tubular: atrophy.
 - Interstitial: fibrosis.
- It is the end – stage of other types of GN.
- It presents with: **CRF & ESRD**.

replacment therapy only sy-p th

End stage renal disease

or dialysis or transplant

5

White Knight Love

CLINICAL PRESENTATIONS

- GN may present with one or more of the following clinical presentations:

1. Nephrotic syndrome.
2. Nephritic syndrome (Acute GN).
3. Urinary abnormalities: e.g. hematuria, proteinuria.
4. ARF: (over days to weeks).
5. Rapidly progressive RF: (over weeks to months).
6. CRF: (over months to years).
7. HYPERTENSION.

(renin → HTN)
 ٥٥ ٥٥ ٥٥ ٥٥ ٥٥ ٥٥

INVESTIGATIONS

1. Urine analysis.

RBCs in IgA
 RBCs
 casts

2. Blood investigations:

e.g.

- CBC
- Lipid profile. Hyperlipidemia in nephrotic
- Plasma proteins. ↓
- Electrolytes. ↑K except tubular disorders nephrotic

3. Renal function tests.

urea
 Creatinine
 Cr. clearance

4. Renal imaging:

e.g.

- Abdominal ultrasonography.

5. Renal biopsy.

كشور
 Patho

6. Investigations for the cause:

e.g.

- Hepatitis markers: for HBV, HCV.
- Serology: for SLE..
- Blood glucose: for DM.

لو
 U, Cr. ↑
 لازم حتماً يكون
 Cr. clearance ↓
 abnormal
 لازم بيظهر بدي

Oral

المفروضه في عيانه
 Bilat. small contracted
 Kidneys

6
 الا في ٥ حالات
 - Angioidosis
 - DM

Oral emergency & PSGN

NEPHRITIC SYNDROME

"ACUTE GN"

DEFINITION

- A disease characterized by the ACUTE ONSET of:

days → weeks

1. HEMATURIA.
2. PROTEINURIA (Sub – nephrotic).
3. OLIGURIA (& ARF). ^{severe oliguria}
4. OEDEMA.
5. HYPERTENSION.

clinically essentially
investigating
acute disease
لا يشخص بـ biopsy

oliguria
ألم حادة
↓ GFR

لأنه يفرس ودية
due to proliferation

لا تنزل بول لانه تنزل RBCs

ETIOLOGY

I. PRIMARY

(Idiopathic & pathology is confined to kidney)

II. SECONDARY

(a part of multisystem disorder)

1. Infections:

• Bacterial:

Post – Streptococcal,

SBE.

• Viral:

HBV,

HCV.

2. Immune:

SLE, PAN, HSP, GPS, EMC.

(GPS) أشر

Abs (shy imm. complex)

Abs complex

الغلبة

no HIV (chronic)
no schistosoma

PATHOLOGY

"Proliferative GN"

1. Diffuse:

A) Membrano – proliferative GN.

B) Mesangio – proliferative GN.

C) Diffuse – proliferative GN.

الوحدة
& also causes nephrotic

التفسير

- ① Prolif. واحد
- ② Extra & intra cap. prolif. الوحدة

2. Focal:

"IgA nephropathy or Berger's disease".

أشهر
سبب لـ
GN
على القصور

أشهر حاد
nephritic
up to 100%
v. acute

Q: post streptococcal sequelae:
 1 R.F.
 2 GN
 3 HSP
 4 Erythema nodosum
 5 R.A.
 8

ACUTE POST - STREPTOCOCCAL GN (PSGN)

ETIOLOGY

- Organism: Nephritogenic strains of group A β - hemolytic streptococci.
- Site of infection: Pharynx or skin.
Pharyngitis *Cellulitis*
- Incubation period: 1 - 3 weeks.
- Age: Young (usually below 20).
- Sex: Males > Females (2 : 1).

PATHOGENESIS

- Immunological injury is due to:

- Deposition of **IMMUNE COMPLEXES** in the glomeruli.
GN due to infection of the kidney
sub endothelial sub epithelial

PATHOLOGY

- Features of: Diffuse proliferative GN.
- Electron microscope: SUBEPITHELIAL IMMUNE DEPOSITS "HUMPS".

Projections due to deposition

Triad -
 - Shlept Ag
 - Antistreptolysin
 - Complement

CLINICAL PICTURE

"ACUTE NEPHRITIC SYNDROME"

1. HEMATURIA.
2. PROTEINURIA (Sub - nephrotic).
3. OLIGURIA (& ARF).
4. OEDEMA.
5. HYPERTENSION.

1. HEMATURIA:

○ PATHOGENESIS:

- Injury to the glomerular capillary wall.

○ CLINICAL FEATURES:

- It occurs in the form of smoky or dark urine, HOWEVER, frank hematuria may occur.

أكثر واحد منه
Hematuria
IgA (Bergers)

also
CSF
TB meningitis

cloudy = (RBCs) not seen grossly
seen only by microscope

2. PROTEINURIA:

= non-nephrotic
(Sub-nephrotic)

نephrotic لا ترى
< 3.5 gm / 24 h

○ PATHOGENESIS:

- Injury to the glomerular capillary wall.

○ CLINICAL FEATURES:

- It is usually detected in urine analysis: (< 3.5 gm / 24 h).

oral لا ترى
heavy Effacement
leaky thickening
is not the case here

3. OLIGURIA: = anuria

(& ARF)

○ PATHOGENESIS: $\downarrow \downarrow$ GFR

- ① - Obstruction of Glomerular capillary lumen by the proliferative lesion:

1. Infiltration of: glomerulus by inflammatory cells (neutrophils & monocytes).
2. Proliferation of: resident glomerular cells.

- ② - Intra-renal VC due to local imbalances of VC & VD substances:

1. VC substances: Leukotrienes & PAF.
2. VD substances: Nitric oxide & Prostacyclin.

as injury → body auto-regulation
to reduce work on injured glomeruli

○ CLINICAL FEATURES:

- Acute onset of oliguria (< 400 ml / day) & ARF: over days to weeks.

also
- mediator = B.A.
- Stroke (evolution)
- meningitis

4. OEDEMA:

○ PATHOGENESIS:

- $\downarrow \downarrow$ GFR. → ↓ excretion of fluid → hypervolemia
- $\uparrow \uparrow$ reabsorption of: salt & water. → inj → renin → Aldosterone

↑ intra vascular pr.
→ ↑ filtration 3rd

○ CLINICAL FEATURES:

- Onset: Acute.
- Site: Peri-orbital region & face (early), may become generalised (late).

L.L.
U.L.
abd
heart
generalized anasarca

as caps of eye
V. weak

nephrotic: chronic: pshuna & hyponatremia
nephritic: Acute: موروقة

WhiteKnightLove

Not cause edema as this less is mild & connected by liver
nephrotic heavy phos
Edema HTN

current & acid
also mediator = B.A.
Anti-LT
Acute GN

Acute
starts eyelid

Oral

Acute unilateral edema
DVT
Acute Bilat L.L. edema

- nephrotic
- allergic (angio-neurotic)

death causes

- ARF
- HTV encephalopathy
- Acute HF → Acute pulm. edema

HTV emergency

Target organ damage

10

5. HYPERTENSION:

○ PATHOGENESIS:

- ↓↓ GFR.
- ↑↑ reabsorption of: salt & water.
- Generalised VC: due to stimulation of **RAAS**.

inj → Renin → Aldosterone
AT₂ → 40 times catecholamine

○ CLINICAL FEATURES:

- Severe Hypertension may pass to: **Hypertensive encephalopathy**.
- Associated CVS manifestations: [due to hypervolemia]
Heart failure: may pass to → **Acute pulmonary oedema**.

6. GENERAL MANIFESTATIONS: "3 P"

- **P**rexia: + headache, malaise, anorexia, nausea, vomiting.
- **P**ain: in both loins. *التهاب الكلى (Bilat) always*
- **P**allor: due to anemia.

damage (↓ erythropoietin)
if ARF (uremic anemia)
if hematuria (bloody)

INVESTIGATIONS

1. Urine analysis:

- Volume: oliguria (< 400 ml / day).
- Aspect: smoky or dark urine, *RBCs* However frank hematuria may occur.
- Specific gravity: increased. *↓ fluid or ↑ solute (phs)*
- Proteins: proteinuria (Sub-nephrotic < 3.5 gm / 24 h).

• MICROSCOPIC EXAMINATION:

- Cells: **Red B Cs** (many & dysmorphic)
- Casts: **Red cell casts**

as injured by injured endothelium

The most important investigation

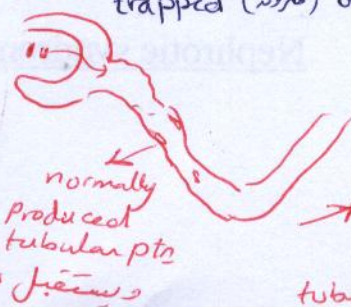
أزول أنت بروتين تحليل عادي
(if) Alb: - +++
أزول 24 hours albumin

2. Blood investigations:

- CBC: Anemia & high ESR. *inflamm.*
- Lipid profile: Normal.
- Plasma proteins: Normal.
- Electrolytes: ↑ K (may be present).

الصبي لا يحب أن RBCs في تحليل البول

= Cylindrical structure: mixture of RBCs trapped (متركة) on tubular ptn Matrix



nephrotic is lipid (waxy) cast

White Knight Love

tubular ptn

$\frac{\text{volume}}{\text{time}} \rightarrow \text{amount of plasma cleared from creatinine / min. time}$

N: 120-125 ml/min

11

3. Renal function tests:

- Impaired glomerular function: $\downarrow$ creatinine clearance (a good estimate of GFR).
- Blood urea & serum creatinine: may be elevated.

4. Renal imaging:

- Abdominal ultrasonography: mild swelling of the kidneys.

5. Renal biopsy:

- It is usually not needed for the diagnosis.
acute

6. Investigations for the cause:

- Streptococcal antibody tests: High ASO titre, Positive ASZ test.
streptococcus
 $\hookrightarrow$ 400 : Adult
 $\hookrightarrow$ 200
or 250 : child

PROGNOSIS

"Better in children than in adults"

- Over 90 % of cases: Complete recovery usually in few weeks.
- About 5 % of cases: RPGN $\rightarrow$ renal failure in weeks to months.
- Few cases: (days) Death from ARF, Hypertensive encephalopathy, APE.

DIFFERENTIAL DIAGNOSIS

- From causes of:

- Nephritic syndrome. *PSGN*

- Hematuria.

- Proteinuria. *2eg <*

- Oliguria & ARF. *2eg <*

- Oedema.

- Secondary hypertension. *2eg <*

- From: Nephrotic syndrome.

انسح
nephrotic

hepatic
nephrotic
allergic
cardiac
acute

20y HTN
Acute
& chronic
GN
& chronic
Pyelonephritis

TREATMENT

A. GENERAL MEASURES:

1. Rest: Complete bed rest.

2. Diet:

- **Salt:** restriction. (as has salt & H₂O retention)
- **Fluid:** restriction in marked oliguria & oedema, allowed when diuresis starts.
- **Potassium:** restriction in case of hyperkalemia. (لوفال مايجبت بول خالص)
- **Protein:** restriction in case of renal failure. (already urea ↑)
- **Fats:** restriction is preferred because they are nauseating.
- **CHO:** no restriction.

in Liver failure
الغذاء انك تبجج فيه
! لا لو الحياه دخلت H₂O
اصح Pte تقام

B. SPECIFIC MEASURES:

1. Antibiotics: Crystalline penicillin, 1 million U / 6 hours (1 week).

2. Symptomatic ttt: e.g.

- Hypertension: Anti-hypertensive drugs, e.g. Hydralazine. (Methyl dopa Loop)
- Oedema: Diuretics, e.g. Furosemide. (not give Ksping)

H.F. digitalis (digitoxin) → فلوكون
يقطع الوريد

- Spironolactone
- ARBs

3. TTT of complications: e.g.

- ARF. dialysis only → لا يفتح
الوريد
التي
transplantation
- Hypertensive encephalopathy. (استرجع كالك)
- Acute pulmonary oedema. furosemide morphine O₂

NEPHROTIC SYNDROME

DEFINITION

- A clinical syndrome characterized by:

1. PROTEINURIA (Heavy) (nephrotic > 3.5 gm / 24 h).
2. HYPOPROTEINEMIA.
3. OEDEMA.
4. HYPERLIPIDEMIA.
5. LIPIDURIA.

- Heavy proteinuria: is the primary abnormality.
- Other features: occur secondary to heavy proteinuria.

ETIOLOGY

I. PRIMARY

(Idiopathic & pathology is confined to kidney)

- all non proliferative
- Minimal change GN: *the most common cause in: CHILDREN.*
 - Membranous GN: *the most common cause in: ADULTS.*
 - Focal segmental glomerulosclerosis (FSGS).
 - Membranoproliferative GN.
- all proliferative →

II. SECONDARY

(a part of multisystem disorder)

1. Infections:

- Bacterial: SBE, Syphilis.
- Viral: HBV, HCV, HIV.
- Parasitic: Schistosoma.

~~PSGN~~ → acute GN
(nephrotic is chronic)

- 14
GPS → acute
2. **Immune:** SLE, PAN, HSP, EMC. (most common causes)
3. **Iatrogenic:** Gold, Penicillamine, Captopril.
4. **METABOLIC:** DM, Amyloidosis. (Kw syndrome stage 2)
5. **MALIGNANCY:** MM, Lymphoma (Hodgkin's).
6. **MISCELLANEOUS:**
- Hereditary: Alport's syndrome, Sickle cell disease.
 - Toxic: Heroin.
- هو ان يخل انه يقاتل
proteinuria
لكن هذا بسبب
idiocyncrasy
في بعض الناس
و من غير ان يكون
عشوائي هاجم الناس به العلاج
عنه

CLINICAL PICTURE

1. PROTEINURIA (Heavy) (Nephrotic):

massive leak of ptn thru disturbed GBM due to either:

① effacement of foot processes (loss of charge & widening of pores)

② diffuse leaky thickening of GBM (widening pores)

PATHOGENESIS:

- Injury to the glomerular capillary wall.

CLINICAL FEATURES:

- It may be one of 2 types:

a) Selective proteinuria: (mild pathology)

- Urine: proteins of low MW ONLY, e.g. albumin.
- Renal pathology: mild.
- Prognosis: good.

b) Non-selective proteinuria: (severe pathology)

- Urine: proteins of low & high MW.
- Renal pathology: marked.
- Prognosis: poor.

- Loss of protein in urine will lead to:

a) Hypoproteinemia.

< albumin only — (selective)
albumin + Globulin — (non selective)

b) Tubular damage: which may progress to CRF

due to heavy proteinuria (injurious on tubules) ← oral

tubular defect

heavy proteinuria
mild not > 2gm

edema

لانه مستحق في بعض الحالات
فالتصغير بين هراتي يدي

فقدان الكبريت في بروتين

سريع

↑ infections

CRF

selective or non → edema
 edema → nephrotic
 15

2. HYPOPROTEINEMIA:

oral
 ptn
 & d/c

○ PATHOGENESIS:

- Heavy proteinuria. exceeding liver capacity to compensate

○ CLINICAL FEATURES:

- Selective & non [
 - Albumin → Generalized oedema (↓ oncotic pr.)
 - Immunoglobulins → Increased susceptibility to infections.
 - Vit. D-binding globulin → Osteomalacia & rickets (Adults) (child)
 - Transferrin → Microcytic hypochromic anemia (iron resistant).
 - Antithrombin - III → Hypercoagulability & Thromboembolism.

nephrotic
 mild loss
 liver
 بيسوف

Loss of:

transudation

oral

لأنك لها إعتبات Fe
 هينزل دهر مع Transferrin
 له إعتبات دائما بيقع
 فلازم تخالج cause الكبد

3. OEDEMA:

selective
 non

○ PATHOGENESIS:

- initiating - Hypoalbuminemia: ↓ oncotic pressure, the most important factor.
- aggravating - Underfill Theory: Hypovolemia stimulates RAAS & stimulates ↑ ADH.

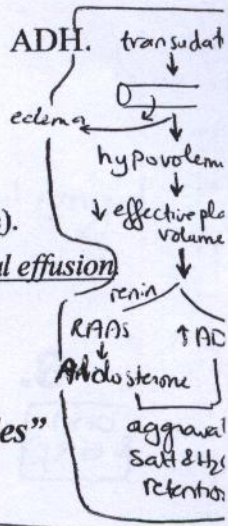
○ CLINICAL FEATURES:

- Onset: Gradual.
- Site: Peri-orbital region & face (early), may become generalised (late).
- Course: Generalized anasarca with ascites, pleural effusion, pericardial effusion
- Character: Soft, pitting. (myxedema - non pitting (MPS))

for nephrotic
 acute
 لأنه بيقع
 إعتبات خايف
 ناليناه بيقع فيه

as cap permeability ↑ (weak wall)
 anasarca

if ascitic threshold reached (sgn)



4. HYPERLIPIDEMIA

"Increased cholesterol & triglycerides"

○ PATHOGENESIS:

- Increased hepatic synthesis of lipids. → due to
- Decreased removal of lipids. → loss of apolipoprotein in urine → mobilizes & catabolize lipids from liver

GRF

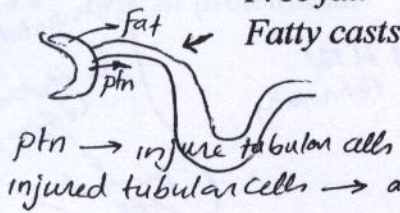
○ CLINICAL FEATURES:

- Complications of: ATHEROSCLEROSIS. (شرح) - CAD - CVD - PAD
- Xanthomata. yellowish plaques - eyelids

5. LIPIDURIA

- In urine, excess lipids & lipoproteins will pass as: as ↑ Blood & GBM

- Free fat.
- Fatty casts (degenerated tubular epithelial cells containing fats).



asymptomatic except if very ↑↑ → frothy urine

White Knight Love

6. Hypokalemia: ↓K

○ PATHOGENESIS:

- Decreased intake:
- Decreased absorption:
- Increased aldosterone:
- Effect of drugs:

Long standing
Anorexia, N, V (Congestive gastropathy)
Oedema of the intestinal mucosa (Long standing Congestive enteropathy)
Secondary hyperaldosteronism. (underfill theory)
Diuretics & Corticosteroids → ↑ K loss

○ CLINICAL FEATURES:

- Refer to: **Kidney.**

انسج

thiazide furosemide
not treated by
K-sparing
as V-weak
فالي فالي
thiazide furosemide
⊕ K supplement

as most common
pathogenesis is immunological

7. Hypocalcemia:

○ PATHOGENESIS:

- Loss of Albumin: will ↓ the albumin-bound Ca carried by this albumin.
- Loss of Vitamin D-binding globulin

○ CLINICAL FEATURES:

- It RARELY results in TETANY because the Ionizable Ca is normal.

except if ↓ vit D

8. Other manifestations:

Oral
& GRF

1. Pallor: due to anemia.

2. GIT: Anorexia, N, V.

3. Renal: Progression to: (CRF & secondary HTN) or (ARF).

○ CRF: due to progressive tubular damage by proteinuria.

○ ARF: due to severe hypovolemia or renal vein thrombosis.
(Severe ↓ GFR)

4. CVS: Accelerated CVD (e.g. CAD & Stroke), due to:

- Hyperlipidemia.
- Hypercoagulability.

↑ **Atherosclerosis**

- Oral
why HTN
- ① Renal damage
 - ② 2ry Hyperaldosteronism
 - ③ H₂O by catals
 - ④ Atherosclerosis

9. Manifestations of the cause:

- e.g. SLE, DM.

انسج

Q young
20-40
auto
immune
GN

associated HTN
retinopathy

Heavy proteinuria
↓ oncotic pr.
transudation
↓ hypovolemia (↓ GFR)
prerenal ARF
White Knight Love

INVESTIGATIONS

1. Urine analysis:

- **Volume:** normal. (if ARF: oliguria) (if CRF: polyuria)
- **Aspect:** frothy. (loss of FAT)
- **Specific gravity:** increased. (loss of p_H & fat)

أهم
ملاحظة
تسخدم

- **Proteins:** heavy proteinuria (nephrotic > 3.5 gm / 24 h).
Proteinuria may be: selective or non-selective.

albumin
only

albumin
& others (globulins)

- **MICROSCOPIC EXAMINATION:**

- **Casts:** **Fatty** casts.

2. Blood investigations:

- **CBC:** Anemia.
- **Lipid profile:** Increased cholesterol & triglycerides.
- **Plasma proteins:** Decreased (mainly, albumin). → always - selective & non
- **Electrolytes:** ↓ K & ↓ Decreased total Ca. despite normal ionized Ca⁺⁺

3. Renal function tests:

- **Normal:** except if renal failure occurs.

4. Renal biopsy:

- It will show the pathology.

أنسجة كلية

5. Investigations for the cause:

- e.g. SLE, DM.
(serology) Bld glucose

PROGNOSIS

"It depends on several factors"

- **Pathology:** it is best in minimal change GN.
- **Age:** it is better in children than in adults.
- **Type of proteinuria:** it is better in selective than non-selective proteinuria.

DIFFERENTIAL DIAGNOSIS

From causes of:

- Nephrotic syndrome. اكتب الاسباب
- Proteinuria. اكتب دواعي
- Oedema. اكتب Cardiac
liver
nutritional
- Hyperlipidemia. اكتب

لا يكتب
allergic (Acute)

From: Nephritic syndrome. اكتب المربع

From: Myxoedema. non pitting
Apathetic
Cold intolerance
abn. thyroid fn.

TREATMENT

A. TTT of the cause:

1. PRIMARY (autoimm)

Minimal change GN:

Prednisone:

ابتدا - 2 mg / Kg / day:
maintenance 1 mg / Kg / day:

for 4 weeks, then:

for 4 weeks, then:

over 4 weeks. if no response to cortisoid

Gradual withdrawal:

Immunosuppressive drugs:

- 2 mg / Kg / day:

for 8 weeks.

- In corticosteroid - resistant cases.

"Add Cyclophosphamide"

Membranous GN:

FSGS:

Membranoproliferative GN:

Corticosteroids
Immunosuppressive drugs

2. SECONDARY:

e.g. SLE, DM.

Cortisoid
+ immunosuppressant

Proper control + 111 kw

علاج
Diabetic
nephropathy
لا يكتب علاج
immunosuppressant

White Knight Love

B. SYMPTOMATIC TTT:

1. Hypoproteinemia:

- DIET: High protein.
- ALBUMIN: IV salt-free albumin.

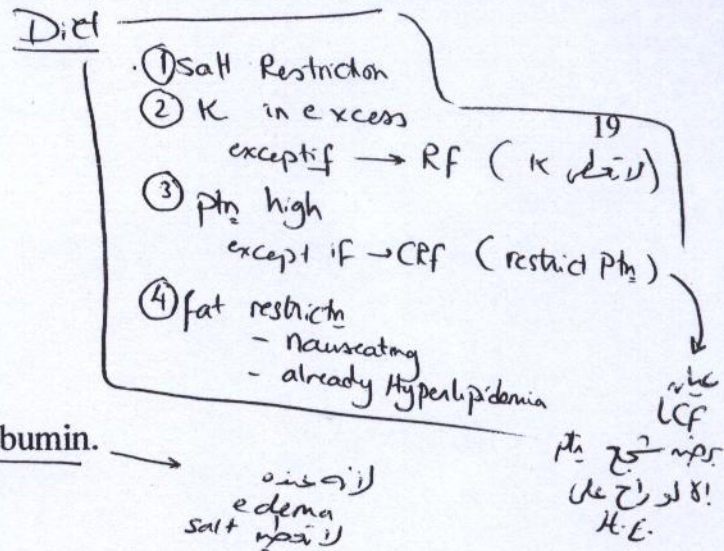
2. Oedema:

- DIET: High protein + salt restriction. → to ↑ Oncotic pr.
- DIURETICS:

سبب و نه بطنی سبب - K-sparing: spironolactone.
 - Non K-sparing: furosemide or thiazides (add K).

3. TTT of complications: e.g.

- Infections. proper control by Antibiotics
- Anemia. vit, B12 transfusion
- Hyperlipidemia. statin or fibrate (cholesterol) (TG)



INTERSTITIAL NEPHRITIS

DEFINITION

- A group of disorders that affect the renal interstitium & the renal tubules.

tubulo interstitial tissue

ETIOLOGY

I. Acute interstitial nephritis: "3 I"

1. Iatrogenic: [Allergic, drug hypersensitivity]
 - NSAIDs. *analgesic nephropathy*
 - Antibiotics: *Methicillin, Sulphonamides.*
 - Diuretics: *Thiazides, Frusemide.*
2. Infection: *acute pyelonephritis.*
3. Immunological: *SLE.*
4. Irradiation.

نازل كمان
eosinophils

II. Chronic interstitial nephritis:

1. Iatrogenic:
 - NSAIDs: prolonged use & abuse (*analgesic nephropathy*).
2. Infection: *chronic pyelonephritis & renal TB.*
3. Immunological: *SLE.*
4. Irradiation.
5. METABOLIC: *DM, gout, nephrocalcinosis.*
6. TOXIC: *Lead, mercury.*
7. NEOPLASTIC: *MM.*
8. OBSTRUCTIVE UROPATHY.

CLINICAL PICTURE

1. Tubular abnormalities: e.g. Fanconi syndrome, RTA.

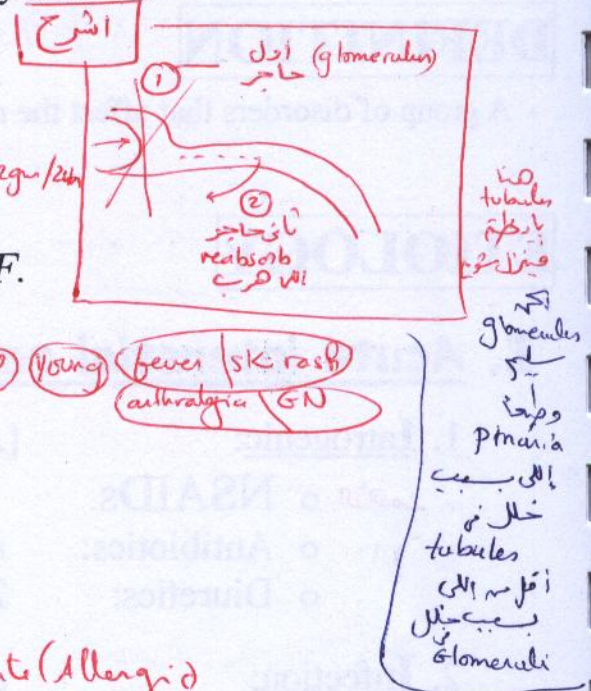
2. Anemia.

3. Proteinuria: (tubular proteinuria) usually mild. $< 2\text{g}/24\text{h}$

4. RENAL FAILURE: ARF or CRF.

5. Features of the cause: e.g. SLE. ♀ (Young) fever / skin rash

GN IN



INVESTIGATIONS

1. Urine analysis:

- Proteinuria, hematuria, eosinophils.

2. Isothenuria. fixed specific gravity

Concentration & dilution of urine failure (as its fn of tubules is here) are affected

3. Renal function tests:

- Impaired:

if renal failure occurs.

4. Renal biopsy:

- It will show the pathology.

5. Investigations for the cause:

e.g. SLE.

Anti ds, Anti sm specific
Anti Nuclear antibody sensitive

TREATMENT

1. Treatment of the cause:

e.g. SLE.

2. Treatment of renal failure:

Dialysis or transplantation.

(chronic)

TUBULAR NEPHROPATHIES

FANCONI SYNDROME

also
hereditary
aplastic
anemia
(Fanconi)

DEFINITION

- It is a disorder in which the proximal tubular function of the kidney is impaired, resulting in decreased reabsorption of electrolytes and nutrients back into the bloodstream.

ETIOLOGY

1. Hereditary

- Isolated.
- Wilson's disease. $\downarrow$ ceruloplasmin $\rightarrow$ $\uparrow$ Cu = tissue & urine
- Cystinosis. inborn error of cysteine metabolism

2. Acquired

- Multiple myeloma. Bence Jones ptn (plasmacells)
 - Drugs: expired tetracyclins.
- damaging on tubules $\rightarrow$ corneal opacity, tubular damage

MANIFESTATIONS

1. Metabolic acidosis. } failure to absorb $\leftarrow \begin{matrix} HCO_3 \\ K \end{matrix}$]
2. Hypokalemia. } الأنسجة المزدروعة]
3. Renal glycosuria. despite it doesn't reach 180 (threshold)] الأنسجة من عارفة ترجع]
4. Polyuria, polydipsia, dehydration. (osmotic)
5. Aminoaciduria.
6. Phosphaturia $\rightarrow$ rickets (in children) or osteomalacia (in adults).

TREATMENT

1. Fluid & electrolyte replacement. $\downarrow K$
2. TTT of the cause. — penicillamine for wilson

RENAL TUBULAR ACIDOSIS

DEFINITION

- It is a medical condition that involves an accumulation of acid in the body due to: failure of the kidneys to appropriately acidify the urine.

tubules

metabolic
acidosis

failure to secrete H^+ ions
" " to reabsorb HCO_3^-
of tubules

ETIOLOGY

1. Hereditary

- Isolated.
- Wilson's disease.
- Cystinosis.

2. Acquired

- Multiple myeloma.
- Drugs:

analgesic nephropathy

amphotericin B.

also
in I.E. (fungal)
also
direct
destructive
H.A.

TYPES

1. TYPE I: [Distal RTA]

- Metabolic acidosis.
- Hypokalemia.
- Nephrocalcinosis & renal stones.

diffuse calcification - parenchyma

Hyperpara
Ca²⁺ in
urine 20-50 mg/day

2. TYPE II: [Proximal RTA]

- Isolated: *loss of HCO_3^- only.*
- Part of Fanconi syndrome: *generalized proximal tubular dysfunction.*

HCO_3^- , K, G

TREATMENT

1. Fluid & electrolyte replacement.
2. TTT of the cause.

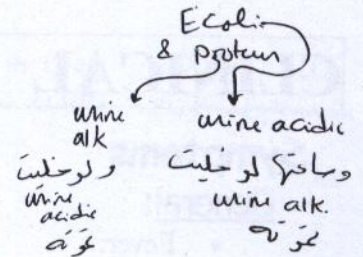
ACUTE PYELONEPHRITIS

Inflammation of pelvis & Parenchyma of Kid

ETIOLOGY

A. Causative organism:

1. Gram negative bacilli: 90 % of the cases
 - E. coli: 80 % of the cases.
 - Klebsiella, Proteus, Pseudomonas: 10 % of the cases.
2. Gram positive cocci: Staph. aureus, Strept. fecalis.
3. Others: RARE fungi, chlamydia, viruses.



B. Routes of infection:

1. Ascending infection: "the most common"
 - In cases of lower UTI.
2. Hematogenous route: "not common"
 - In cases of BACTEREMIA, e.g. SBE, Pneumonia.
3. Lymphatic route.

C. Predisposing factors:

- Local**
1. Impaired urine flow: "Urinary tract obstruction" O.U. (stasis)
 - Renal pelvis: stone, tumour.
 - Ureters: stone, tumour, stricture.
 - Bladder: bladder neck obstruction, neurogenic bladder.
 - Urethra: stone, enlarged prostate, stricture.
- Systemic**
2. Impaired immunity:
 - **A**IDS.
 - **B**ad general condition: general debility, old age, malnutrition.
 - **C**orticosteroids.
 - **D**iabetes mellitus. $\rightsquigarrow$ AMP $\rightsquigarrow$ ARF
 3. Introduction of infection:
 - **C**atheterization, Cystoscopy, Surgery of the bladder.
 - **C**ystitis: "Honeymoon cystitis" frequent intercourse $\rightarrow$ more in σ (as long urethra) more commonly injured

also
Pneumonia
& TB

PATHOLOGY

- ❖ Inflammation: of the mucous membrane of the kidney.
- ❖ Abscesses: Multiple small abscesses are present with streaks of pus.

COMPLICATIONS

1. Perinephric abscess.
2. Pyonephrosis.
3. Chronic pyelonephritis.
4. Septicemia.



Blood

↑ stone
metabolic
DM
Gout
hyperpar

CLINICAL PICTURE

Symptoms

General:

- Fever:

Abdominal:

- Pain:
- Urinary symptoms:
- Nausea & vomiting.

acute with rigors.

polyuria & oliguria

acute pain in the loin radiating to the groin.

frequency, dysuria, pyuria, strangury.

due to pain → reflex pyrospasm

* Painful micturition & sense of incomplete evacuation followed by persistent desire to micturate

relaxation of urethral sphincter

Signs

General:

- Fever:

acute with pallor. Toxic inhibition of B.M.

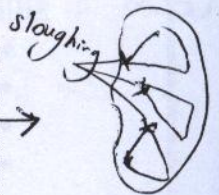
Abdominal:

- Tenderness:

in the renal angle.

Acute Necrotizing Papillitis

- A severe form of infection leading to papillary necrosis. tubules damage
- It is most common in: Diabetes mellitus
- It presents with severe acute pyelonephritis & severe hematuria.
- It is complicated by: ARF.



INVESTIGATIONS

1. Urine analysis:

- Volume: normal.
- Aspect: normal OR turbid. (pus cells)
- pH: ACIDIC in E. coli (most cases), ALKALINE in proteus.
- Proteins: mild proteinuria is present.
- Microscopic examination:
 - Cells: Many pus cells, Some RBCs, Organisms.
 - Casts: White cell casts.

(if ANP & ARF : oliguria)

epith. damage

B. cub

Ascarbic Acid

as infect

False due to local production from cells of tubules

White Knight Love

2. Urine culture & sensitivity:

- Bacterial count more than 100,000 / ml: denotes infection.
- Bacterial count less than 100,000 / ml: denotes contamination.

3. Blood picture:

- Polymorphnuclear leucocytosis with shift to the left.

↑ immature (staf = band cells)

4. Renal function tests:

- Normal (except in ANP).

5. Renal imaging:

- e.g. ^{21VP} PUT & ultrasonography, for detection of:
 - Predisposing factors: e.g. stones.
 - Complications: e.g. perinephric abscess.

if in Kid : appears in u/s
if in ureter : not appear in u/s

DIFFERENTIAL DIAGNOSIS

1. Fever with rigors. *eg of other infections & feveriggers*
2. Abdominal pain. *eg of surgical medical causes*
3. Pyuria, *e.g. cystitis.*

TREATMENT**I. GENERAL TREATMENT**

- Rest in bed.
- Excessive fluid intake.
- Analgesics & antipyretics.

II. SPECIFIC TREATMENT**1. Antibiotics:**

"for 1 - 2 weeks"

- Before the results of urine C & S: give a broad spectrum antibiotic, e.g.
 - Quinolones: *Ciprofloxacin* 500 mg bid orally.
 - Tetracyclines: *Doxycycline* 100 mg bid orally.
- Resistant & Recurrent cases: give antibiotic according to urine C & S:
 - For E. coli: *Ciprofloxacin* 500 mg bid orally, or *Nitrofurantoin* 100 mg / 8 h.
 - For Pseudomonas: *Carbimicillin* 1 gm / 6 h IV.
 - For other Gm - ve bacilli: *Gentamycin* 80 mg / 8 h IV or IM.

2. Change the pH of the urine:

- If the urine is acidic: give *sodium bicarbonate*.
- If the urine is alkaline: give *ascorbic acid*.

III. CORRECTION OF PREDISPOSING FACTORS. *eg surgical removal of stone.***IV. TREATMENT OF COMPLICATIONS.**

eg surgical drainage of PN Abscess

CHRONIC PYELONEPHRITIS

علاج
مضاد حيوي

DEFINITION

- **CHRONIC** interstitial nephritis resulting from **RECURRENT** bacterial infection of the kidney.
- The disease is either: *unilateral* or *bilateral asymmetrical*.

ETIOLOGY

- **RECURRENT** acute pyelonephritis

unilat Acute (stone mostly unilat.)

PATHOLOGY

1. Macroscopic appearance:

- Kidney:

SMALL with irregular surface & adherent capsule.
fibrosis of chronicity

2. Microscopic appearance:

- Tubules: atrophic.
- Glomeruli: periglomerular fibrosis.
- Interstitial tissue: chronic inflammatory cells & excessive fibrosis.

CLINICAL PICTURE

- The patient may present with *one* OR *more* of the following manifestations:

1. **Recurrent** attacks of acute pyelonephritis.
2. **Recurrent** attacks of general non-specific symptoms, e.g. *back pain*, *fatigue*, *anorexia*.
3. **Chronic** anemia — *ACD uremia (Rf)*
4. **Chronic** renal failure.
5. **Secondary hypertension** — *damage → reni*
6. **Symptomless** proteinuria.

*2ry H TN
due to
Chronic
not
acute
PN*

INVESTIGATIONS

1. Urine analysis:

- **Proteins:** mild proteinuria is present (less than 3.5 gm / 24 hours).
- **Microscopic examination:**
 - Cells: Pus cells, RBCs, Organisms.
 - Casts: **White cell casts.**
- **In late stages:** features similar to CRF.

*CRF
1st common cause DM
C/PN (stones due to β)*

RENAL TUBERCULOSIS

ETIOLOGY

- **Organism:** Mycobacterium TB.
- **Infection:** Hematogenous spread from a primary infection.
- **Predisposing factor:** Immunodeficiency, e.g. AIDS.

Q magit :
diagnosis
+ Ht 26
clerk

CLINICAL PICTURE

Symptoms

General:

- TB toxemia: night sweats, night fever, loss of weight, loss of appetite.

Abdominal:

- Pain: pain in the loin radiating to the groin.
- Urinary symptoms: frequency, dysuria, pyuria, strangury, hematuria.

Signs

General:

- Fever: with pallor, toxic face, loss of weight.

Abdominal:

- Tenderness: in the renal angle.

erosion of BV
by caseating
granuloma

lung: Apical fibrosis or Cavitation

Patients may have active TB in other parts of body.

COMPLICATIONS

1. **CRF:**

in bilateral cases. Common

2. **Calcification.**

3. **Secondary** hypertension.

damage → ↑ renin

4. **Secondary** amyloidosis.

chronic → amyloid

5. **Spread:**

e.g. genital TB.

6. **Stricture** of the ureter:

leading to obstructive uropathy.
fibrosis

2.1, 2
amyloidosis

INVESTIGATIONS

1. Urine analysis:

- ~~Sterile~~ pyuria.
- Hematuria.
- Urine is subjected to:
 - Staining: using ZN stain.
 - C & S: using Lowenstein medium & BACTEC.
 - Animal inoculation: using guinea pig.

Culture is -ve

أهم
نقطة
هنا

2. Blood investigations:

- Anemia.
- ESR: *markedly elevated.*

↑ ESR

TB
Malig
autoimm
anemia

3. Renal imaging:

PUT & ultrasonography & IVP, for detection of:

- Calcification.
- Stricture of the ureter.

4. Renal biopsy:

- It will show the pathology: refer to "Chest".

5. PCR: (Polymerase Chain Reaction)

- For detection of mycobacterial DNA.

استرج
Cavate
Cells
Calcified

6. Tuberculin test:

- It is usually positive.

TREATMENT

I. GENERAL TREATMENT

- Rest in bed.
- Proper nutrition.

II. SPECIFIC TREATMENT

- Anti - TB ttt: refer to "Chest".

III. TREATMENT OF COMPLICATIONS

- Surgery: for stricture of the ureter.

Anti TB ttt

*كامل
الجرعة*

وقت حاسي كوتيزون

Q: Causes of ARF = Causes of oliguria
 Q: management
 Q: investigation

ACUTE RENAL FAILURE

DEFINITION

- A disease characterized by **acute** rapid decline of the GFR over hours or days:
 - It is usually associated with oliguria: urine volume less than 400 ml / day.
 - It is frequently reversible.

recovery (esp if acute tubular necrosis)

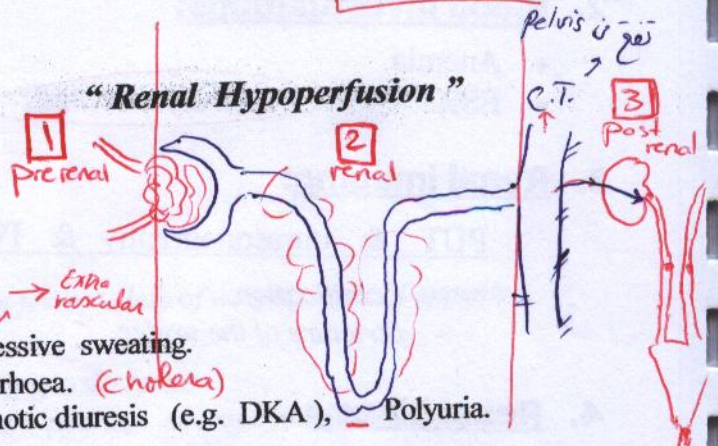
ETIOLOGY = etio of oliguria

I. PRE-RENAL: Kid. normal

55 %

A. Decrease in blood volume:

1. SEVERE HEMORRHAGE.
2. SEVERE HYPOALBUMINEMIA.
3. Skin fluid loss: Burns, Excessive sweating.
4. GIT fluid loss: Vomiting, Diarrhoea. (Cholera)
5. Renal fluid loss: Diuretics, Osmotic diuresis (e.g. DKA), Polyuria.



B. Decrease in the renal blood flow:

1. Shock: cardiogenic shock & septic shock.
2. Severe heart failure: and other causes of ↓ CO as sustained arrhythmias.
3. Severe liver failure: hepatorenal syndrome.
4. Severe renal ischemia: bilateral renal artery thrombosis or embolism. (causes of hypercoag. state)

II. INTRINSIC RENAL: 40 % "Diseased renal parenchyma"

A. Diseases of the tubules:

1. Acute tubular necrosis (ATN): "the most common cause"

- a) Ischemic: Prolonged reduction of the renal blood flow, ie, Persistence of the pre-renal causes (any cause).
- b) Toxic:
 - Exogenous: aminoglycosides, cyclosporin, heavy metals.
 - Endogenous: hyperuricemia, hemolysis.

2. Acute necrotizing papillitis.

3. Acute interstitial nephritis.

NSAID

allergy (eosinophil)

act by both

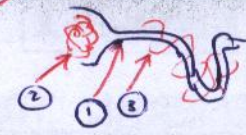
- Toxic injury
- obstruction of tubules

gentamycin (I.E.)

streptomycin (anti TB)

Neomycin (Hepatic encephalopathy)

White Knight Love



Oral emergency

→ acute massive skeletal muscle destruction

① Trauma (crush syndrome)

② rarely: statin: usually myositis & rarely rhabdomyolysis

③ Heat stroke

④ Extensive repeated convulsions

Obstruction

⑤ Acute alcoholic intoxication

⑥ Severe hypokalemia

↓K → MS destruction

hyperkalemia tubular obstruction

in hyperuricemia.

4. Tubular obstruction:

a) Crystals as uric acid

b) Proteins:

- Hemoglobin: toxic in
- Myoglobin: toxic in
- Myeloma proteins: in

hemolytic crisis.

rhabdomyolysis. Trauma forms

multiple myeloma (plasma cells: 80%)

Bence Jones

injury obstruction

never Hb 32 in urine as:

- Trapped in blood by hemopexin & haptoglobin
- reabsorbed by tubules

B. Diseases of the glomeruli:

1. Membrano-proliferative GN.
2. Mesangio-proliferative GN.
3. Diffuse proliferative GN → acute GN.
4. Crescentic GN → RP GN

e.g. Goodpasture's syndrome.

C. Vascular diseases:

1. Malignant hypertension. → target organ damage
2. Eclampsia.
3. Vasculitis: SLE, PAN.
4. HUS, TTP, DIC. Endoth. damage intravascular coagulation

MAHA

III. POST-RENAL:

5% "obstruction of the urine flow"

1. Bilateral ureteric obstruction: e.g. stones.
2. Unilateral ureteric obstruction: in a single kidney.
3. Obstruction of the urethra or the bladder neck.

Kidney is normal

Pathogenesis of ATN

- ATN passes through 3 phases:

1. Oliguric phase: [1-2 weeks] "Injury of the tubules with ↓ GFR due to":

1. LEAK: (backleak) of the glomerular filtrate through the injured tubules back to the blood.
2. Intrarenal VC: due to release of VC mediators from injured endothelial cells.
3. Obstruction: of the tubules by the injured tubular debris. (granular cast)
- All the above: will result in decline of GFR → decreased urine volume.

2. Polyuric phase: "partial recovery of the tubules with excessive diuresis due to:"

- Excretion of retained salt & water.
- Delayed recovery of tubular reabsorption compared to glomerular filtration.
- All the above: will result in increased urine volume.

3. Recovery phase: "complete recovery of the tubules"

- The completely recovering tubules will regain their power to fully reabsorb water and to concentrate the urine, leading to normal urine volume.

Pathology of ATN

- | | | |
|--------------|----------------------|--|
| 1. Ischemic: | - Distribution: | Patchy necrosis of the whole tubule. |
| | - Basement membrane: | Damaged. |
| 2. Toxic: | - Distribution: | Diffuse necrosis of the proximal tubule. |
| | - Basement membrane: | Intact. |

CLINICAL PICTURE

I. Manifestations of the oliguric phase:

1. Oliguria:

- Volume: Less than 400 ml of urine per day. Absolute anuria is more common in post-renal obstruction. *O.U.*
- Duration: It usually lasts few days to few weeks. *↑ CVP*

2. Hypervolemia:

"Overhydration"

- Circulatory congestion: Congested NVs, pulmonary congestion & APE. *RT heart* *LT heart*
- Coma: *renin (renal damage)* Secondary hypertension & oedema. *LL* [DCL] Convulsions, Confusion, Coma.

3. Hyperkalemia:

"see later". (failed excretion)

4. Acidosis:

- Rapid deep respiration: Kussmaul's breathing. (failed excretion of H^+ , failed reabs. of HCO_3^-)

5. Uremic syndrome:

"clinical features of uremia"

- A:** Anorexia, nausea, vomiting, hiccough. *gastitis* *phenic N. irritate*

Anemia. *Uremia* *very thrombocytopenia*

Asthenia. *V. rare*

- B:** Bleeding tendency, GIT hemorrhage. *if gastritis → G. ulcer* *due to bleeding tendency*
Pruritus.
Pericarditis & Pleurisy.

- C:** Convulsions, Confusion, Coma. (toxin)

II. Manifestations of the polyuric phase:

- Volume: urine volume reaches up to 5-10 L/day.
- Effect: hypotension, dehydration, electrolyte loss. *hyponatremia*

III. Manifestations of the recovery phase:

- Volume: urine volume returns to normal.
- Effect: clinical manifestations improve.

INVESTIGATIONS

1. Urine analysis:

- **Volume:** Less than 400 ml / day, may be absolute anuria.
- **Aspect:** Dark. *injury*
- **Specific gravity:** Low fixed "in the range of 1010" in cases of ATN. *Fixed: failed tubules to concentrate or dilute urine*
- **Proteins:** mild proteinuria is present. *Low: ↓ volume reabsorption of solutes*
- **Microscopic examination:**
 - Cells: Epithelial cells & RBCs. *tubular failure to reabsorb*
 - Casts: **Granular casts.** *injury*

2. Blood investigations:

- Anemia. *of uremia*
- Increased K *↑* Increased P *↑* *[Failure to secrete]*
- Decreased Na *↓* Decreased Ca *↓* *[Failure to reabsorb]*
- Decreased HCO₃ *↓* Decreased pH *↓*

granular cytoplasm of injured epithelial cells (diagnostic to ATN)

3. Renal function tests:

- There is marked impairment of the renal functions:
- Increased: creatinine, urea, uric acid.
- Decreased: creatinine clearance: an early good estimate of ↓ GFR.

4. Renal imaging:

PUT & Ultrasonography:

- Post-renal obstruction: e.g. stones.

5. Renal biopsy:

indicated in:

- Persistent ARF: more than 4 weeks.

*القاعدة أن لو رجم
acute
عنوع
biopsy*

6. Investigations for the cause.

*eg DKA
Sugar and acetone in urine*

7. ECG:

may show features of Hyperkalemia.

Pericarditis [diffuse ST elevation]

TREATMENT

1. Exclusion:

- Exclusion of urinary retention: by bladder catheterization.
- Exclusion of post-renal obstruction: by investigations, & obstruction should be treated.

2. Treatment of the cause:

- Pre-renal causes: IV fluids for hypovolemia, or blood transfusion for hemorrhage.
- Toxic agents: should be stopped. *eg stop aminoglycoside*

3. Induction of diuresis:

"treatment of the oliguric phase"

- Frusemide: 2 - 10 mg / Kg IV.
- Dopamine: 2 ug / Kg / min. IV infusion. *+++ RBF*
- Mannitol: 500 ml of 25 % solution IV infusion.

*dopamine renal dose
work on Kid
diuretic*

*dopamine high dose
work on CVS
the in
VC
White Knight Love*

4. Conservative treatment: (-Symptom H)

a) Treatment of Hypervolemia:

- Salt & fluid restriction.

b) Treatment of Hyperkalemia:

- See later. *أهمية الشح*

c) Treatment of acidosis:

- Sodium bicarbonate: by IV infusion in severe cases (pH less than 7.2).

d) Diet treatment:

- Salt & fluid restriction: to correct Hypervolemia.
- Potassium restriction: to correct Hyperkalemia.
- Protein restriction: to lower the blood urea (0.5 gm / Kg / day).
- Carbohydrates: ↑ allowed in excess to decrease protein catabolism.
- Fats: avoided because they are nauseating.

e) Symptomatic treatment:

- Hypertension, pulmonary oedema, anemia, vomiting, etc.....
- Hyperphosphatemia: Phosphate restriction & give Calcium carbonate.

f) Treatment during the polyuric phase:

- Adequate fluid intake to avoid hypovolemia & dehydration.
- Adequate replacement of electrolyte losses. Na to avoid hyponatremia.

5. Dialysis treatment:

♥ Indications:

A) Laboratory:

1. Serum creatinine: below 10 mg / dl.
2. Blood urea: above 200 mg / dl.
3. Serum K: above 7 or 6.5 mEq / L.
4. Bicarbonate: below 10 mEq / L.

B) Clinical:

1. Coma or convulsions.
2. Pulmonary oedema.
3. Pericarditis. *لأنه مناعها أنه بسحوم*
4. Severe bleeding. *زادت أدوية*

♥ Methods:

1. Hemodialysis.
2. Peritoneal dialysis.

CHRONIC RENAL FAILURE (CRF)

DEFINITION

"Multi system disease"

- A disease characterized by gradual progressive deterioration of renal functions over months or years:

- It is **IRREVERSIBLE**.
- The clinical manifestations of end-stage renal failure are: "uremia or uremic syndrome".

Urine in Blood → [Chronic prolonged Blood Poisoning]

ETIOLOGY

Chronic
Acute

[the most common 2 causes are: **DM** & **CP**]

Chronic
Pyelonephritis
stones

1. INFECTIONS

- Chronic Pyelonephritis. *
- Bilateral renal TB.

2. IMMUNE

- Glomerulonephritis: especially chronic GN.
- Goodpasture's syndrome. → Acute → when recurrent
- SLE, PAN, Henoch - Schonlein Purpura.
- Scleroderma.

3. METABOLIC

- Diabetic nephropathy, *
- Amyloidosis, *
- gouty nephropathy, *
- nephrocalcinosis. hyper para

KW Syndrome

4. MALIGNANT

- Multiple myeloma.

5. TOXIC

- Analgesic nephropathy. NSAID → chronic interstitial nephritis
- Irradiation nephropathy. (R.T.)
- Heavy metals: lead, mercury.

6. VASCULAR

- Hypertension: especially malignant hypertension.
- Recurrent renal infarctions. [SCA] → damage vs.

7. OBSTRUCTIVE

- Bilateral hydronephrosis. (Bilat stones)

8. CONGENITAL

- Polycystic kidney. Bilat
- Renal hypoplasia.

Kidney enlarged

- DM
- Amyloid
- Hydronephr
- PCK

PATHOLOGY

ماہر لائزہ و سیکشن تیز فی حدائی (کریسٹ) ARF

1. Macroscopic appearance:

- Kidney: small with irregular surface & adherent capsule.

2. Microscopic appearance:

- Interstitial tissue: excessive fibrosis.
 - Tubules: atrophic.
 - Glomeruli: atrophic.
- Chronic GN
وہ لائزہ کی وجہ سے
Chronic

CLINICAL PICTURE

The uremic syndrome results from both: glomerular & tubular dysfunctions:

1. WATER, ELECTROLYTE & ACID-BASE DISORDERS

1) Water:

- Polyuria:** due to:
 - Osmotic diuresis: due to increased solute load per nephron.
 - Failure of tubules to respond to ADH.
- Oliguria:** up to anuria
 - May occur in late stages.
- Isothenuria:**
 - Low fixed specific gravity "in the range of 1010".

Decreased ability to concentrate or dilute urine:

- Increased water intake → hypervolemia & overhydration.
- Decreased water intake → hypovolemia & dehydration.

2) Sodium: 2 types

- Sodium retention:**
 - It occurs in most patients especially in glomerular diseases.
 - Cause: it is due to inability of the kidney to excrete Na load.
 - Effect: hypervolemia, hypertension, pulmonary congestion, oedema.
- Sodium loss:** "salt-losing nephropathy"
 - It occurs more commonly in patients with tubular diseases.
 - Cause: it is due to inability of the tubules to reabsorb Na.
 - Effect: hypotension & muscle cramps.

3) Potassium:

- Increased serum K due to extracellular shift of K.

4) Phosphorus & Calcium:

- Increased P & decreased Ca.

5) Metabolic acidosis:

- Due to failure to secrete H & to form HCO_3^- .

Kussmaul's

Filtration (excretion)

reabsorption

2. CARDIOVASCULAR MANIFESTATIONS

- 1) Hypertension: due to salt & water retention or increased rennin.
- 2) Arterial disease: accelerated atherosclerosis due to hyperlipidemia. *loss of apolipo ptn (↓ mobilization)*
- 3) Pericarditis: dry or hemorrhagic. *(bleeding tendency)*

3. RESPIRATORY MANIFESTATIONS

- 1) Rapid deep breathing: Kussmaul's breathing, due to acidosis. *also DKA*
- 2) Recurrent chest infections: due to impaired immunity.
- 3) Pulmonary oedema: due to hypervolemia. *salt retaining (glomerular)*
- 4) Pleurisy. *dry or hagic*
- 5) Uremic asthma. *toxins (urea) → irritates Bronchial tree (spasm)*
- 6) Ulcers: in the respiratory tract → hemoptysis.

4. GIT MANIFESTATIONS

- 1) Mouth: ammoniacal odour + stomatitis. *ammoniacal (urea) (ammonia)*
- 2) Tongue: *dry* + coated. *gastroduodenal (G. ulcers)*
- 3) Stomach: anorexia + nausea + vomiting + hiccough + hematemesis.
- 4) Intestine: constipation + bleeding. *intestinal ulcers (lytotoxic)*
- 5) Uremic dysentery: diarrhoea + tenesmus with blood & mucus in stools due to: ulcers in the mucus membrane of the colon & rectum. *(oral)*
- 6) Increased incidence of HCV in patients on regular hemodialysis.

5. RENAL MANIFESTATIONS

- See before: Water disorders.

6. NEUROLOGICAL MANIFESTATIONS

- 1) Apathy. *poor cerebration (toxins)*
- 2) Asterixis. *rarely (Hx = Liver & Resp)*
- 3) Psychiatric symptoms: irritability, depression. *(neurotransmitter disturbance)*
- 4) Peripheral neuropathy: & myopathy. *(degeneration by toxins)*
- 5) Convulsions.
- 6) Confusion & coma: uremic coma or uremic encephalopathy.
- 7) Dialysis dementia: usually due to aluminium intoxication.
- 8) Drowsiness, fatigue, headache & lack of concentration. *(VD by toxins)*

7. GENITAL MANIFESTATIONS

- Males: impotence & infertility.

- Females: amenorrhea & infertility.

phosphate binding
Aluminium gel
P
Aluminium
toxication
Blood
P
Hyperphosphatemia
Al
Aluminium
toxication
Blood
P
Hyperphosphatemia
Al

Aluminium
toxication

Al
Hyperphosphatemia

- 1 angular stomatitis
- 2 Gastric ulcer (hematemesis)
- 3 Small int. " (hematemesis)
- 4 large int. " (melena)

dementia: deteriorated
brain. in cognitive
fn. (تدهور في
الوظائف العقلية)

الدماغ المتدهور
تدهور في الوظائف العقلية
! إلى سوء وظائفه (المرضى)
(fluid rich in Al.)

White Knight Love

8. MUSCULOSKELETAL MANIFESTATIONS

- 1) Renal osteodystrophy: "due to secondary hyperparathyroidism"
- Osteomalacia, osteoporosis, osteitis fibrosa cystica. *Haversian's lacunae*
 - Bone pains & pathological fractures may occur.
- 2) Myopathy. *toxins* → gradual prolonged degeneration (+) ↑K
- 3) Muscle cramps: due to decreased Na. (tubular)

damage
Bone is
weak
kidney

hepatic
osteodystrophy
in PBC
due to
↓ vit D
(obstructed
dig.)

9. SKIN MANIFESTATIONS

- Pallor:** & earthy colour of the skin.
- Pruritus.** *toxic + dark complexion*
- Pigmentation:** *pale urine* *pallor also skin*
- Uremic frost:** deposits of urea crystals.
- Dehydrated skin.** *failure kidney to excrete* *urochrome*

toxins
hyperparathyroidism
if infected
(local
pruritus)

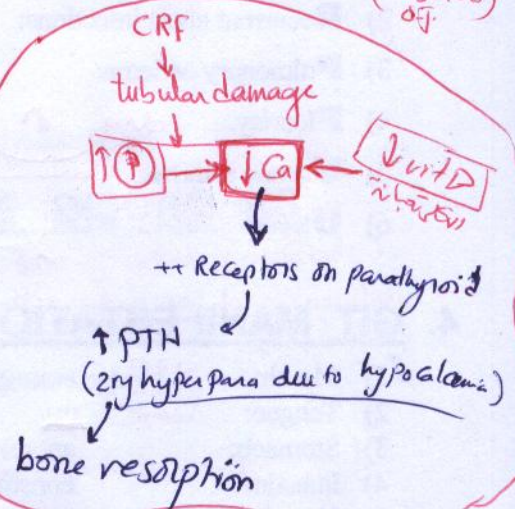
10. BLOOD MANIFESTATIONS

1) Anemia:

- ↓ production of EPO.
- ↓ iron.
- ↓ vitamins (folic acid, vitamin B₁₂).
- ↓ life span of RBCs (hemolysis).
- HYPERPARATHYROIDISM.

نقص
اللون
البي
نقص
الهيموجلوبين
الخلايا
الحمراء

Similar to pancytopenia
انخفاض في [الخلايا الحمراء] و [الخلايا البيضاء] و [الخلايا الصلبة]



لأنه
أوقف
العمل
في
Symptomatic Ht of
↑ P
↓ Ca
↓ vit D
لأنه
Cause

جذب الكالسيوم Antacid / Ca, vit D

2) Bleeding tendency: due to:

- Platelet dysfunction: (Coated platelets) *Thrombocytopenia*
- Toxic inhibition of the clotting factors.

"the most important factor"

3) Increased liability to infections: due to:

- Lymphocyte & phagocyte dysfunction: "the most important factor"
- Increased exposure to infection in patients on dialysis.

Coated
by toxins

11. EYE MANIFESTATIONS

- Uremic retinitis.

12. METABOLIC MANIFESTATIONS

- Disturbance in CHO metabolism may be one of 2 types:
 - Early: Hypoglycemia due to decreased renal clearance of insulin. *down regulation of R*
 - Later: Hyperglycemia (IGT or secondary diabetes) due to insulin resistance.

13. MANIFESTATIONS DUE TO DIALYSIS COMPLICATIONS

- See later.

اس طرح ہے

INVESTIGATIONS

1. Urine analysis:

- **Volume:** Polyuria, about 3–4 L/day. Oliguria occurs in late stages. *ESRD*
- **Aspect:** Pale.
- **Specific gravity:** Low fixed "in the range of 1010".
- **Proteins:** mild proteinuria is present.
- **Microscopic examination:**
 - Casts: **Broad casts** & Granular casts. *large granular cast*
inj. of tubules

2. Blood investigations:

- Anemia.
- Increased K
- Variable Na
- Decreased HCO_3
- Increased P.
- Decreased Ca.
- Decreased pH.

الظا ہے
ARF

3. Renal function tests:

- There is marked impairment of the renal functions:
 - Increased: creatinine, urea, uric acid.
 - Decreased: creatinine clearance: an early good estimate of $\downarrow$ GFR.

4. Renal imaging:

- e.g. PUT & ultrasonography, for detection of:
 - The cause & the renal size.

5. Renal biopsy:

- Will reveal the pathology.

میکروسکوپیک

6. Investigations for the cause.

eg DM: Blood Glucose $\text{FGS} > 126$
 $\text{HbA}_{1c} > 200$

7. ECG:

may show features of Hyperkalemia.

C.P. الی

Bilat. small contracted Kid. except

- Amyloidosis
- Hydronephrosis
- PCK
- DM

ہیپلینوسس
فبروسس

لأني باستخدام CUP ← to monitor I.V. Volume

♥ Complications:

1. Hemodialysis: Dialysis dementia, air embolism.
2. Peritoneal dialysis: Peritonitis, abdominal hernias.
3. Both types: HCV, AIDS & other infections.

2. RENAL TRANSPLANTATION:

- It is the ONLY curative ttt for irreversible renal failure.

National Kidney Foundation

فسيحة

Cr. clearance N: 120 - 125 ml/min

CRF	stage 1	: CRF e ↓ Cr. clearance (minimal)	90 - 120
	" 2	: - - - - -	(mild)
	" 3	: - - - - -	60 - 90
	" 4	: - - - - -	(moderate)
ESRD	" 5	: - - - - -	30 - 60
			(severe)
			10 - 30
			< 10

أحمر شائع
للزراعة
Kid
liver
Heart
lung
Pancreas x x

AUGUST

(13 hours each day: Pleeceeeeeeeeeeeeeeeeease)

30 / 7 – 3 / 8: 5 days VACATION (No book whatsoever).

4 / 8 – 8 / 9: STUDY: (35 days): as follows:

- Medicine : Surgery = 8 hours : 5 hours / day.
- Both should be preferably studied together each day.
- I NEED FROM YOU THE FOLLOWING IN AUGUST:

- KIDNEY:	ALL	(2 days):	4 – 5
- INFECTIONS & RHEUMATOLOGY:	ALL	(2 days):	6 – 7
- CHEST:	ALL	(4 days):	8 – 11
- BLOOD:	ALL	(3 days):	12 – 14
- NEUROLOGY:	ALL	(6 days):	15 – 20
- ENDOCRINE:	ALL	(4 days):	21 – 24
- GIT & LIVER:	ALL	(3 days):	25 – 27
- Clinical, paraclinical		(3 days):	28 – 30
- Skin, psychiatry		(1 day):	31
- CARDIOLOGY:	ALL	(7 days):	1 / 9 – 7 / 9

CARDIOLOGY: will be studied starting from day 1 in September because we will start our revision in September by CARDIOLOGY (see later).

SEPTEMBER

(16 hours each day: Pleeceeeeeeeeeeeeeeeeease)

- From: 1st September you will start studying Medicine HARDLY.
- This is the period of four REVISION of MEDICINE.
- Revision will start on: 8th September (more or less) by Cardiology which should be studied hardly starting from 1st of September (ALL CARDIOLOGY).
- Study of Medicine in September will proceed according to the timetable of Revision.
- The timetable of Revision will be provided to you soon.

Remember always:	It is always a question of: “To be or not to be.”
Remember always:	It is the time of a very strong will & the real desire for perfect achievement.
Remember always:	It is now or never that you will be able to reach your goal in life.
Remember always:	You still have time but don't let it escape away from you.
Remember always:	Taking rest in the coming period is a big luxury that you cannot afford.
Remember always:	I will always be there for you if you need me.
Remember always:	I trust you & you will do an excellent performance.

Good luck Sherif EL Hawary

- In September: Study should be: Medicine : Surgery = 80 % : 20 %.
- Both should be preferably studied together each day in the ratio of: 80 % : 20 %.
- You come to the Revision as if you are coming to the final exam.

OCTOBER

(16 hours each day: Pleeeeeeeeeeeeeeeeeeeease)

- This is the period of REVISION OF SURGERY.

- Medicine : Surgery = 20 % : 80 %.
- Both should be preferably studied together each day in the ratio of: 20 % : 80 %.

THE FINAL COUNT DOWN

(17 hours each day: Pleeeeeeeeeeeeeeeeeeeease)

- 21 days should be left for pure Medicine before the exam.
- This is the period of 100 % MEDICINE.
- First part: (5 days) *paper 1*.
- Second part: (9 days) *paper 2*.
- Third part: (7 days) *paper 1*.

NB: Some supplementary papers will be provided to you upon return during the revision...

Remember always:	It is always a question of: "To be or not to be."
Remember always:	It is the time of a very strong will & the real desire for perfect achievement.
Remember always:	It is now or never that you will be able to reach your goal in life.
Remember always:	You still have time but don't let it escape away from you.
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Remember always:	I trust you & you will do an excellent performance.

Good luck Sherif EL Hawary

في القاع SQ
or Oral 1000

43

HYPERKALEMIA

SQ
Oral emergency

ETIOLOGY

1. INCREASED INTAKE

"Iatrogenic"

- Excessive therapy with IV potassium.
- Excessive transfusion of stored blood.

overcorrection of hypokalemia

المفرط
وانت بتدق تقسيم في 2-7 ملل

2. DECREASED LOSS

- Renal failure: **Acute** & **Chronic**.
- Adrenal failure: Addison's disease.
- Drugs: Potassium-sparing diuretic (e.g. spironolactone), ACE inhibitor (e.g. captopril).

↓ cortisol → ↑ K

لأن الكورتيزول يحفز

aldosterone antagonist

3. EXTRACELLULAR SHIFT

- Acidosis.
- Insulin deficiency. **DKA** $\xleftrightarrow{H^+}$ $\xleftarrow{K^+}$
- Tissue damage (**lysis**): hemolysis, rhabdomyolysis, tumour lysis syndrome.

↑ K in Intracellular

K, P, U.A in Ht leukemia

CLINICAL PICTURE

- 2 Hs
- Heart: Bradycardia, Heart block,
 - Muscle: Myopathy, Muscle weakness,

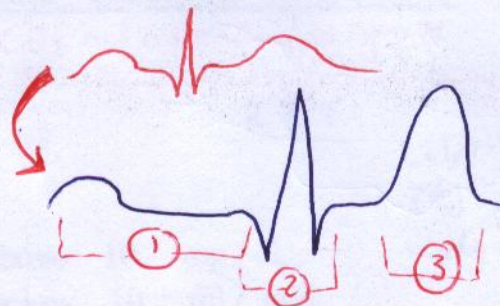
Cardiac arrest in diastole.
Muscle paralysis.

INVESTIGATIONS

1. **Serum K:**
- Increased (normal: 3.5 - 5 mEq/L).

2. ECG:

- P-R interval: prolonged.
- QRS complex: widened.
- T wave: tall & peaked (hyperacute T wave).
- Bradycardia, Heart block.



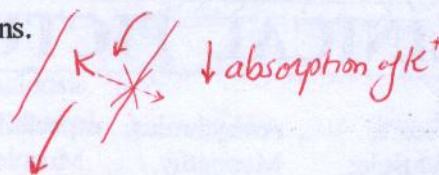
3. Investigations for the cause:

- e.g. elevated urea & creatinine in acute & chronic renal failure.

TREATMENT

1. DECREASED INTAKE

- **D**iet: Restriction of potassium in diet.
- **D**rugs: Restriction of drugs that $\uparrow$ K, e.g. *spironolactone* & *captopril*.
- **D**ecrease absorption: Resins, as cation-exchange resins.



2. INCREASED LOSS

- **D**iuretics: e.g. frusemide.
- **D**ialysis: in severe cases (K above 7 mEq/L).

3. INTRACELLULAR SHIFT

- Correction of acidosis: NaHCO_3 100 mEq IV.
- Insulin + glucose: Regular insulin 10 U + glucose 25 gm IV infusion.

تصحيح DKA كنه لا يرفع acidosis بل يخفضه
وكانه انشيت insulin بتولن PK الى K
خلال علاج كنه انشيت K

هنا لا يوجد
hypoglycemia

4. ANTAGONIZE THE EFFECT OF HYPERKALEMIA ON HEART

- Calcium gluconate: 10 ml of 10% solution over 10 minutes.

also tetany & anaphylactic shock

HYPOKALEMIA

SC
oral emergency

ETIOLOGY

1. DECREASED INTAKE

- IV fluid therapy: deficient in potassium.
- Diet: deficient in potassium.
- Decreased absorption: malabsorption syndrome.

الكورتيزون يحسب دمج

2. INCREASED LOSS

- Renal loss:**
 - Diuresis: Non potassium-sparing diuretics (loop & thiazides), osmotic diuresis.
 - Cushing's syndrome & Conn's syndrome & Corticosteroid therapy.
- GIT loss:**
 - Diarrhoea: especially secretory diarrhoea. *Cholera*
 - Vomiting.

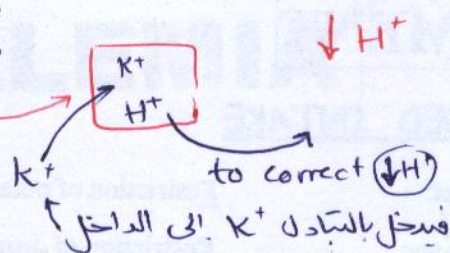
in DKA
due to Extracell. shift $\rightarrow$ K $\uparrow$
but total is normal
or $\downarrow$
(diuresis)

- Excessive tapping of ascites.** (tapping will lead to evacuate peritoneal ascites & this fluid will be re-compensated from Blood $\downarrow$ K)

aggravation $\downarrow$ K
لذلك يمنع H.E. لا يجرى
White Knight Love

3. INTRACELLULAR SHIFT

- Alkalosis.
- Insulin therapy. *لو كثير*
- Diet rich in carbohydrates.



CLINICAL PICTURE

1. Heart: Arrhythmias, especially in patients receiving digitalis.
2. Muscle: Myopathy, Muscle weakness, Muscle paralysis.
3. GIT: Constipation, distension, paralytic ileus.
4. Kidney: Damage of the renal tubules.
5. CNS: Tetany due to associated alkalosis.

alkalosis
hypo kalemia

INVESTIGATIONS

1. Serum K:

- Decreased (normal: 3.5 – 5 mEq / L).

2. ECG:

- ST segment: depressed.
- T wave: flat or inverted.
- Prominent U wave.

3. Investigations for the cause:

- e.g. investigations for malabsorption syndrome.

eg: urinary D-xylose test

TREATMENT

1. Potassium replacement:

- In severe cases: IV potassium with careful monitoring.
- In mild cases: oral potassium is preferred to avoid hyperkalemia.

2. Treatment of the cause.

eg repeated vomiting → Anti-emetics

*القاعدة
وعند
ذلك
أولاً
أفعل
↑K*

*severe → الالو
أدوية IV و أفعل
careful monitor*

*as digitalis is
arrhythmogenic
وال K يتنافس
على digitalis
Na-K
atpase
↓
K في الخلية
digitalis
unopposed
on the pump*

*may
reach
rhabdo-
myolysis
↓
↑K
correct K
or even ↑K*

*nephrogenic
OI
tubules
not respond
to ADH*

*also
caused
by - lithium
- hyper calcemia*

*ST
elevate
Acute MI
Prinzmetal
Acute pericard*



diastole

قاعدة ١ . PH of Blood is suitable ratio of $\frac{CO_2}{HCO_3}$
 قاعدة ٢ . $\uparrow \frac{CO_2}{HCO_3} = \text{acidosis}$, $\downarrow \frac{CO_2}{HCO_3} = \text{alkalosis}$
 قاعدة ٣ . metabolic = HCO_3 change , Resp = CO_2 change

قاعدة ٤ . if CO_2 changed HCO_3 is changed to correct
 والعكس صحيح
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METABOLIC ACIDOSIS

ETIOLOGY

1. Increased acid:

a) Increased acid production:

- Endogenous: Diabetic ketoacidosis, Lactic acidosis.
- Exogenous: Salicylates, Methyl alcohol.

b) Decreased acid excretion:

- Renal failure: Acute & Chronic.

2. Decreased alkali:

a) GIT causes:

- Severe diarrhoea.
- Fistula: pancreatic, biliary, intestinal. loss of alk. juices

b) Renal causes:

- Tubular defects: Fanconi syndrome, Renal tubular acidosis.
- Drugs: Spironolactone.

↑ aldosterone antagonist / ↓ aldosterone
 (1) ↑ K⁺ (2) ↓ Na⁺ intracellular

أي شيء منه خلل في PH الدم وأنت المصابي
 e.g. Resp acidosis
 $\uparrow\uparrow\uparrow CO_2$
 $\uparrow HCO_3$
 أنت تتنفس في سرعة عالية جداً
 لكي تكمل HCO_3 تتلاشى زائده كعقوبة
 من Kid. لكي من يكتفي
 أنت يمنع الـ acidosis
 ولا يمكن تلاقى واحد عالي
 والآخر راسل لأنه على واحد
 يحللي أويوطين لازم
 اثنان يحللي أويوطين على

CLINICAL PICTURE

1. Respiratory: Hyperventilation: Kussmaul's breathing (rapid deep breathing).
2. Heart: Myocardial depression. (in COPD → HF)
3. Bone: Decalcification in chronic cases.
4. CNS: Drowsiness & impaired consciousness in severe cases.

(acidosis) ++RC
 → to wash out CO_2
 ratio
 acidosis ↓ cortical neurons

INVESTIGATIONS

1. Low pH of the blood.
2. Low HCO_3 & PCO_2 .
3. Investigations for the cause. ↑ Bld glucose, urine sugar & acetone

also - DKA
 coma

TREATMENT

1. $NaHCO_3$ IV.
2. Treatment of the cause. e.g. DKA → insulin & IV fluids.

Ratio $\frac{HCO_3^-}{P_{H_2O}}$ kidney $\frac{HCO_3^-}{P_{H_2O}}$ $\frac{HCO_3^-}{P_{H_2O}}$

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RESPIRATORY ACIDOSIS

SQ
oral

ETIOLOGY

- All causes of hypercapnic respiratory failure that occur due to hypoventilation: "Refer to chest".

CLINICAL PICTURE

- Features of respiratory failure: "Refer to chest".

type II $\frac{HCO_3^-}{P_{H_2O}}$ ventilation
hypoventilation

INVESTIGATIONS

1. Low pH of the blood.
2. High PCO_2 & HCO_3^- .
3. Investigations for the cause. eg CXR for COPD

TREATMENT

- Treatment of respiratory failure: "Refer to chest".

شرح

METABOLIC ALKALOSIS

ETIOLOGY

1. Increased intake of alkali:

- Excessive ttt with bicarbonate.
- Milk-alkali syndrome in ttt of peptic ulcer.

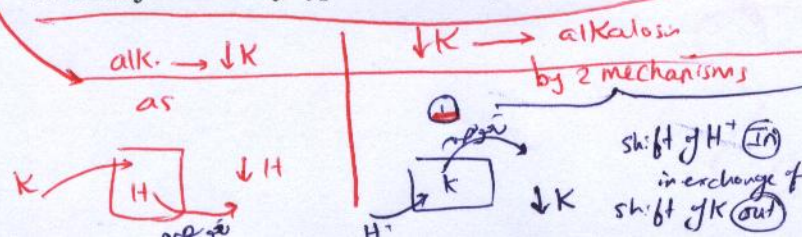
2. Decreased acid:

- a) GIT loss: Vomiting & gastric drainage.
- b) Renal loss: Diuretics esp. loop diuretics, Conn's syndrome, Cushing's syndrome.

3. Hypokalemia.

CLINICAL PICTURE

1. Respiratory: Hypoventilation: shallow slow breathing.
2. Tetany. as alkalosis $\rightarrow$ $\downarrow$ ionized Ca^{++} $[Ca^{++}]$
3. Manifestations of hypokalemia may occur.



over correction of acidosis
- as in ttt of DKA by $HCO_3^- \uparrow$
- as in milk alkali $\rightarrow$ $\uparrow$ Ca $\uparrow$ alk.

hypocholesteremic alkalosis
 Cl^- $\rightarrow$ $\downarrow$ secretion of H^+ ion

$\downarrow$ K $\rightarrow$ upregulation of H^+-K^+ ATPase
 K^+ $\rightarrow$ $\uparrow$ reabs. of K^+ & $\uparrow$ loss of H^+
 $\rightarrow$ alkalosis

WhiteKnightLove

INVESTIGATIONS

1. High pH of the blood.
2. High HCO_3^- & PCO_2
3. Investigations for the cause.

TREATMENT

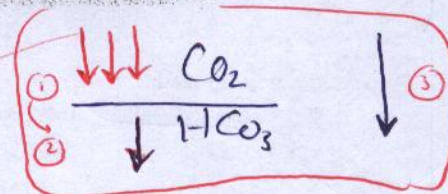
1. Treatment of the cause. *eg correction of ↓ K*
2. Diluted HCL: in severe cases.

RESPIRATORY ALKALOSIS

ETIOLOGY

ALL CAUSES OF HYPERVENTILATION:

e.g.



- Hysteria. *++ RC*
- Head injury or encephalitis.
- Heat stroke.
- Heart failure or pulmonary embolism or pneumonia. *++ periph. Receptors*

*LSHF
J receptors*

chemicals

pulm. R

CLINICAL PICTURE

1. Hyperventilation.
2. Parasthesia. *tingly & numbness*
3. Tetany: *in severe cases.*

INVESTIGATIONS

1. High pH of the blood.
2. Low PCO_2 & HCO_3^- .
3. Investigations for the cause.

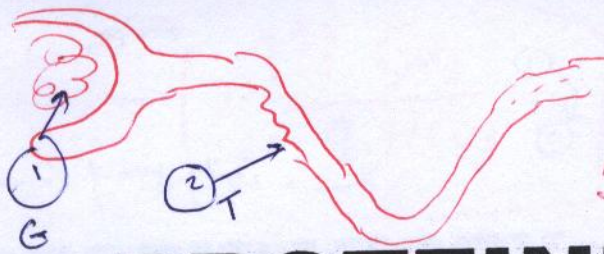
CXR for pneumonia

TREATMENT

1. Treatment of the cause.
2. Hysterical cases: should rebreathe in a paper bag + sedation.

↑ CO2

*hiccuph
↓
↑ CO2 to stop
it
by resp. inhibition*



0-150-g / 24 hr
150-300 microalbuminuria
>300

DM
myeloma
paraneoplastic
Adrenal CA
NSG/Endo

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PROTEINURIA

Q: causes proteinuria

DEFINITION

- Presence of more than 300 mg of proteins in 24-hour urine.

ETIOLOGY

1. Glomerular proteinuria:

- Pathogenesis: increased glomerular permeability.
- Causes: nephritic syndrome & nephrotic syndrome.
- Amount: nephritic syndrome: < 3.5 gm / 24 h, nephrotic syndrome: > 3.5 gm / 24 h.

2. Tubular proteinuria:

- Pathogenesis: decreased reabsorption of the normally filtered proteins.
- Causes: tubular diseases & interstitial nephritis. $\rightarrow$ = tubules = Tubulo interstitial
- Amount: mild proteinuria: < 2 gm / 24 h.

3. Overflow proteinuria:

- Pathogenesis: increased filtration of proteins of low molecular weight.
- Causes: hemoglobinuria, myoglobinuria, Bence-Jones proteinuria. $\rightarrow$ not albumin or globulin
- Amount: variable ranging from: 0.2 - 10 gm / day.

4. Miscellaneous causes:

- Pathogenesis: not clear.
- Causes: HF, Heavy exercise, High fever, Anemia, Convulsions.
- Amount: mild proteinuria: < 2 gm / 24 h.

NB False proteinuria

- It results from loss of protein from the lining of the UT, e.g. INFECTIONS.

P.N.

MICROALBUMINURIA

- Pathogenesis: excretion of small amounts of albumin in urine due to nephropathy.
- Causes: early indicator of diabetic nephropathy. $\rightarrow$ ولو كانت موجودة
- Amount: minimal proteinuria: 150 - 300 mg / 24 h. $\rightarrow$ he is candidate for ACEI

الحمد لله الذي تم بشفاء إيماني

تراجع ٧ و ٨ و ٩ شهر ٩

١٠ - ١٤ / ١٦
شهر ٨ شهر ٩ شهر ١١

المذاكرة تقرأ كثير

الأول الرئيس الصيغة

كل حاجة تتذكر بجانب الأول المهم

• outlines

والعناوين أهم حاجة
وتسبب

• ابدأ و Cases الأول للأهم

• في أهم تذاكر مرفوعة

ICD
الرجاء

الرجوع على كل الكتب في

- لا تنسى should not panic

- أهم حاجة في الساعة ليلة الجمعة اليوم على الأقل ٦ ساعات

- Value ٥.21
عامة
لنقلها الله لا تنسى
- مع هذه دس ممل

- يوم لغاية النتيجة

- الأهم السعارة

- الجارادة الساعة

- حفر أهلا